

## Letters

### Ingestion of shark liver associated with pseudotumour cerebri due to acute hypervitaminosis A

Sir: The syndrome of benign intracranial hypertension or pseudotumour cerebri resulting from hypervitaminosis A was first described by Gerber *et al* in 1954.<sup>1</sup> Acute hypervitaminosis A following the ingestion of polar bear liver is known to occur and arctic explorers have manifested the symptoms of pseudotumour cerebri *viz.* severe headache, vomiting, drowsiness and irritability a few hours after the ingestion of polar bear liver, as reported by Elisha Kane in an anecdotal account of arctic exploration in the 19th century.<sup>2</sup>

Pseudotumour cerebri due to acute hypervitaminosis A associated with the ingestion of shark liver (*Scoliodon* sp.) has not been previously documented although Lonie in 1950 described violent frontal headache, nausea, vomiting, vertigo, drowsiness and irritability in adults following the ingestion of shark liver.<sup>3</sup> Indeed a recent review article on pseudotumour cerebri could not identify a single adequately documented case where acute hypervitaminosis A was the aetiological factor.<sup>4</sup> We therefore report a case of pseudotumour cerebri due to acute hypervitaminosis A following the ingestion of shark liver.

A 25-year-old previously well housewife was transferred from a peripheral coastal hospital to our department with a history of persistent headache, vomiting and diplopia developing over a period of one week following ingestion of a meal of cooked shark liver. All the other family members too who had partaken of this meal had developed those symptoms thus necessitating admission to the local hospital but had recovered with symptomatic treatment within a few days. The persistent nature of our patient's symptoms coupled with the discovery of bilateral papilloedema resulted in her referral to the Neurology Unit for exclusion of a possible intracranial space-occupying lesion. Examination revealed an afebrile, healthy looking, slim, alert female with bilateral florid papilloedema, enlarged blind spots and bilateral partial abducens palsies. There were no other abnormalities. Plain skull radiographs, both posteroanterior and lateral views, were normal there being no evidence of longstanding raised intracranial pressure; the EEG, bilateral carotid

angiography and myodil ventriculography too did not reveal any abnormality (facilities for computed tomography are presently unavailable in Sri Lanka). Analysis of the ventricular cerebrospinal fluid obtained at the time of ventriculography revealed no abnormality. Routine haematology, liver function tests, urine analysis and serum electrolyte estimation including serum calcium were within normal limits. Estimation of serum vitamin A levels using the spectrophotometric method of Neeld and Pearson revealed markedly elevated levels of vitamin A: 177.3 µg/dl (normal range 35–70 µg/dl) while the β-carotene levels were within normal limits: 87.46 µg/dl (normal 50–200 µg/dl).<sup>5</sup> Hepatic vitamin A levels were determined by the fluorometric method of Thompson *et al* on a liver biopsy specimen obtained using the Menghini needle.<sup>6</sup> The hepatic vitamin A content of the patient was 18 mg/100 g liver tissue (fresh weight) while the mean value for healthy adults in Sri Lanka was 10.77 ± 1.2 mg/100 g liver tissue (TMS Atukorala, M Thamotheram, unpublished observations). The patient made an extremely satisfactory recovery over a period of eight weeks and at the time of discharge was asymptomatic; there was no external ophthalmoplegia, blind spots were normal in size and the papilloedema had receded while an EEG revealed no focal or paroxysmal activity. Serum vitamin A levels estimated prior to discharge was within normal limits: 68 µg/dl. The liver biopsy, was not repeated prior to discharge on account of the patient's excellent recovery.

The shark species *Scoliodon* abounds in the coastal waters around the island of Sri Lanka and is frequently consumed by coastal dwellers. The vitamin A content of fish liver in general varies between 2000 and 100,000 IU vitamin A/g, with the shark liver in particular containing one of the highest concentrations of vitamin A.<sup>7</sup> Our patient who consumed approximately 150 g (5 oz) of shark liver would therefore have ingested at least 7,650,000 IU of Vitamin A. It has been estimated that daily ingestion of 100,000 IU or more of vitamin A over a period of a few months is required to produce the pseudotumour cerebri syndrome.<sup>8</sup> Our experience would support this view, although information about this patient's previous vitamin A status was not available. Further supportive evidence of the fact that the origin of this patient's markedly elevated serum vitamin A levels was from an animal source was shown by the fact that her serum β-carotene levels

were not elevated, thus virtually excluding the possibility of the vitamin A being derived from a plant source rich in β-carotene, eg. carrots, mangoes, papaw.

While the mechanism of papilloedema in hypervitaminosis A remains hypothetical, it must be emphasised that it is indistinguishable from the papilloedema due to a brain tumour. Pseudotumour cerebri, therefore remains a diagnosis of exclusion. The above case highlights the need for the physician to be alert to the possibility of acute hypervitaminosis A induced pseudotumour cerebri in areas where shark liver is consumed. Furthermore in cases of pseudotumour cerebri where no obvious aetiological factor can be identified, inquiry into the patients' dietary history may prove worthwhile.

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### References

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- 3 Lonie TC. Excessive vitamin A as a cause of food poisoning. *NZ Med J* 1950;49:680-5.
- 4 Ahlskog JE, O'Neill BP. Pseudotumour cerebri. *Ann Intern Med* 1982;97(2):249-56.
- 5 Neeld JB jr, Pearson WN. Macro and micro methods for the determination of serum vitamin A using trifluoroacetic acid. *J Nutrition* 1963;79:454.
- 6 Thompson JN, Erbody P, Brien R, Murray TK. Fluorometric determination of vitamin A in human blood and liver. *Biochem Med* 1971;5:67-89.
- 7 White A, Handler P, Smith EL. *Principles of Biochemistry*, 5th ed. Tokyo: McGraw-Hill Kogakusha Ltd. 1973;1188.
- 8 Walsh FB, Hoyt WF. *Clinical Neuro-ophthalmology*, 3rd ed. Baltimore: Williams and Wilkins, 1969.

### Thymolipoma in association with late-onset myasthenia gravis.

Sir: The value of steroid treatment and thymectomy in patients with myasthenia gravis without thymomas, especially in